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Maternal programming: Application of a developmental psychopathology perspective

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Abstract

The fetal phase of life has long been recognized as a sensitive period of development. Here we posit that pregnancy represents a simultaneous sensitive period for the adult female with broad and persisting consequences for her health and development, including risk for psychopathology. In this review, we examine the transition to motherhood through the lens of developmental psychopathology. Specifically, we summarize the typical and atypical changes in brain and behavior that characterize the perinatal period. We highlight how the exceptional neuroplasticity exhibited by women during this life phase may account for increased vulnerability for psychopathology. Further, we discuss several modes of signaling that are available to the fetus to affect maternal phenotypes (hormones, motor activity, and gene transfer) and also illustrate how evolutionary perspectives can help explain how and why fetal functions may contribute to maternal psychopathology. The developmental psychopathology perspective has spurred advances in understanding risk and resilience for mental health in many domains. As such, it is surprising that this major epoch in the female life span has yet to benefit fully from similar applications.

The developmental psychopathology perspective provides a valuable framework for understanding mental health disorders and has particularly contributed to improved diagnosis and treatment for children and adolescents (Cicchetti & Toth, 2009; Sroufe, 2013). Given that a central tenant of this approach is focus on characterizing normative development to inform understanding of atypical or aberrant psychology and behavior and that this approach is especially amenable to the investigation of transitional points in development across the life span (Toth & Cicchetti, 2010), it is surprising that one significant developmental epoch in the female life span, that associated with the transition to motherhood, has yet to receive full benefit from this perspective (Bos, 2017; Goodman & Dimidjian, 2012). The dynamic processes of pregnancy and lactation represent a developmental inflection point, one that is arguably the most fundamental and profound in the postnatal female life span. Pregnancy initiates broad and dramatic alterations in maternal anatomy, physiology, and metabolism (Strauss, Barbieri, & Macy Ladd, 2014; Torgersen & Curran, 2006; Williams, 2003). Among these changes is the growth and development of a

new organ, the placenta, that has immune, endocrine, and vascular properties (Petraglia, Florio, Nappi, & Genazzani, 1996). The pregnant woman exhibits endocrine alterations (the scope of which are unrivaled by any other in the postnatal life span), increases in blood volumes and cardiac output, hypercoagulation, insulin resistance, and a shift toward a Th-2 cytokine profile (Greer, 1999; Kuhl, 1991; Poole & Claman, 2004; Torgersen & Curran, 2006). These changes are among those comprising the extensive transformation of maternal physiology necessary to maintain the pregnancy and to prepare the maternal brain for the challenges of motherhood. However, to date, characterization of typical changes in human brain and behavior during pregnancy and the postpartum period are lacking, limiting the ability understand risk for psychopathology that may arise during this sensitive period of development. Nonetheless, here, we begin to address this issue by synthesizing what is known thus far about normative and pathological neuropsychological changes that occur during the peripartum period and how they relate to biological processes during this time.

Maternal Programming: Typical Developmental Trajectories Associated With Motherhood

It has long been recognized that the fetal period represents a sensitive phase in the human life span. However, what is less frequently acknowledged is that pregnancy and the postpartum period represent sensitive periods of neurological development for the mother as well (Glynn & Sandman, 2011). There exists a very well-articulated literature describing maternal changes in brain and behavior in nonhuman animal species, particularly rodents. Leaders in this area of research have documented that as a result of pregnancy, delivery, lactation, and interaction with offspring, females show changes in a range of behavioral domains, such as fear response, aggression, receptiveness to pups, and also domains that more broadly support parental performance including memory and planning (Bridges, 1984; Fahrbach, Morrell, & Pfaff, 1985; Kinsley et al., 1999; Li & Fleming, 2003; Numan & Insel, 2003; Numan, Rosenblatt, & Komisaruk, 1977). The neurological systems that control the development and onset of maternal behavior also are well described (cf. Barrett & Fleming, 2011; Bridges, 2015), and it is established that reproductive history is correlated with neuronal structure, signaling, and neurogenesis (Brummelte, Pawluski, & Galea, 2006; Byrnes, Casey, & Bridges, 2012; Byrnes, Casey, Carini, & Bridges, 2013; Keyser-Marcus et al., 2001; Kinsley et al., 2006; Pfaff, Waters, Khan, Zhang, & Numan, 2011; Shingo et al., 2003).

Despite evidence that most of these neurological changes in rodent mothers persist throughout the life span and may be cumulative (i.e., with additional each litter, the effects are magnified; Gatewood et al., 2005; Lemaire et al., 2006; Love et al., 2005), little is known about typical changes in the human female during this developmental period, although like other species, several physiological alterations implicated in brain function are apparent. For example, in humans and nonhuman primates, such as rodents, the prenatal endocrine milieu appears to set the stage for the onset and development of sensitive and effective maternal behavior (Bardi, French, Ramirez, & Brent, 2004; Feldman, Weller, Zagoory-Sharon, & Levine, 2007; Fleming, Ruble, Krieger, & Wong, 1997; Glynn, Davis, Sandman, & Goldberg, 2016; Maestripieri & Zehr, 1998; Saltzman & Abbott, 2009). Further, the

hormone exposures of pregnancy predict attachment and persisting alterations in maternal behaviors that are detectable at least as late as the end of the first postpartum year (Glynn et al., 2016).

In addition to overtly maternal behaviors, another widely observed biobehavioral alteration coincident with motherhood is downregulation of stress responding, both physiological and behavioral, in humans (de Weerth & Buitelaar, 2005; Glynn, Wadhwa, Dunkel Schetter, Chicz-Demet, & Sandman, 2001) and other animals (Slattery & Neumann, 2008; Vierin & Bouissou, 2001; Wartella et al., 2003). During pregnancy, in humans, responsiveness of the hypothalamic–pituitary–adrenal and sympathetic–adrenal–medullary systems are progressively dampened (Matthews & Rodin, 1992; Nisell, Hjemdahl, Linde, & Lunell, 1985a, 1985b; Schulte, Weisner, & Allolio, 1990), and these changes are similarly observed in psychological responses to stressful challenges (Glynn, Dunkel Schetter, Wadhwa, & Sandman, 2004; Glynn et al., 2001). There is reason to believe that this downregulation of stress responding may serve to protect the mother and fetus from the adverse effects of acute stressors as pregnancy advances; women who do not show this typical change are at increased risk for preterm delivery (Buss et al., 2009; Glynn, Dunkel Schetter, Hobel, & Sandman, 2008). The downregulated stress response then is sustained in the postpartum period by the endocrine profile of lactation (Heinrichs, Neumann, & Ehler, 2002) and also is coupled with enhanced aggression in response to threat, which may allow the mother to more successfully protect and provide for her offspring (Hahn-Holbrook, Holt-Lunstad, Holbrook, Coyne, & Lawson, 2011). Additional capabilities to protect and tend to young successfully that have been documented in humans during pregnancy include deeper nonconscious processing of facial cues (Raz, 2014) and enhanced emotion recognition (Pearson, Lightman, & Evans, 2009).

Unlike the concordance across species in the domain of stress responding and aggression, the patterns in the domain of cognitive function are somewhat less homologous. Among rodent mothers, pregnancy, or manipulation of hormones that simulate the prenatal endocrine milieu, result in enhanced spatial, working, and recognition memory, increased planning abilities, and a reduced latency to prey capture (Kinsley et al., 1999, 2014; Lambert et al., 2005; Macbeth, Gautreaux, & Luine, 2008; Pawluski, Vanderbyl, Ragan, & Galea, 2006). However, in humans, there is little evidence of enhanced function in these domains. Many memory and attentional functions appear to be unaffected by pregnancy in humans (Henry & Rendell, 2007), and furthermore, it has been repeatedly documented that episodic and verbal recall memory are diminished (Glynn, 2010a, 2012; Henry & Rendell, 2007). Much remains to be determined about what precise aspects of cognitive function are altered as a result of the transition to motherhood and also whether or not these alterations persist as they do throughout the life span in other nonhuman animals.

Most recently, researchers have begun to assess the neurological transformations supporting the transition to motherhood in humans (Kim, Leckman, Mayes, Feldman, et al., 2010; Kim, Leckman, Mayes, Newman, et al., 2010; Swain, Lorberbaum, Kose, & Strathearn, 2007; Swain et al., 2008). Motherhood is associated with increased functional connectivity in regions that may subserve sensitive and responsive maternal behavior (Atzil, Hendler, & Feldman, 2011; Atzil et al., 2017; Swain et al., 2017). Further, stronger intrinsic connectivity

(functional connectivity between brain regions assessed with functional magnetic resonance imaging during a resting or task-free state) in the medial-amygdala network is associated with increased dopamine levels within this network, providing some of the first evidence among humans of dopaminergic mechanisms underlying human maternal behavior (Atzil et al., 2017). Most recent are findings documenting change in gray matter volumes from pre- to postpregnancy (Hoekzema et al., 2017). Specifically, changes in gray matter volume are related to maternal attachment and are observable at least 2 years after delivery. These emerging findings highlighting the neurological transformation of the human female, make a compelling case for additional investigation of the neural mechanisms that may underlie both risk and resilience for maternal psychopathology.

Fetal Contributions to Maternal Developmental Trajectories

In 1968, Bell published a seminal paper challenging existing models of the parent–child relationship as unidirectional, underscoring the reciprocal nature of the processes occurring between parent and child and permanently altering the way in which we characterize this unit. In contrast, this bidirectional conceptualization is largely ignored in the consideration of this unique relationship in the context of the prenatal period. While it is widely recognized that maternal signals shape the development of the fetus, it is not as widely acknowledged that this is only one side of the relationship and that fetal and placental signals also may shape the development of the maternal brain and behavior. In order for the fetus to effect change in maternal phenotypes, signals need to be transmitted from the intrauterine compartment into the maternal compartment. Several modes of signaling are available to the fetus and may transmit information across the fetal–maternal threshold. Abundant previous research has explored signal transmission in the opposite direction, but here we uniquely highlight three “languages” in which signals may be communicated from fetus to mother: (a) hormones; (b) motor activity; and (c) gene transfer.

The maternal–fetal endocrine milieu

The placenta is made up of mostly fetal tissue (villous chorion), and these fetal-identity cells (trophoblasts) produce hormones that are secreted into maternal circulation (Cuffe, Holland, Salomon, Rice, & Perkins, 2017; Kliman, 1999; Mastorakos & Ilias, 2003). Although there is understanding of how some fetal endocrine signals may influence the development of maternal behaviors and risk for psychopathology (discussed below), it remains largely unknown whether many hormones that are synthesized from the placenta and released into the maternal compartment are involved in shaping maternal phenotypes. This is the case for chorionic gonadotropin (Braunstein, Rasor, Adler, Danzer, & Wade, 1976), activin (Muttukrishna, Child, Groome, & Ledger, 1997; Petraglia et al., 1994), inhibin (Muttukrishna et al., 1997), relaxin (Eddie et al., 1986; Sakbun, Koay, & Bryant-Greenwood, 1987; Tkachenko, Shchekochikhin, & Schrier, 2014), leptin (Masuzaki et al., 1997), gonadotropin-releasing hormone (Siler-Khodr, Khodr, & Valenzuela, 1984), and human placental lactogen (hPL; Kliman, 1994).

Other hormones that traverse the fetomaternal interface have more complicated derivations. Across many species during gestation, predictable and large increases in estrogens and

progesterone are observed. Human serum estradiol and progesterone levels during pregnancy reflect placental and maternal (ovarian and adrenal) secretion; however, those observed in the maternal circulation are overwhelmingly of placental origin (Diczfalusy & Troen, 1962). Placental estradiol and progesterone are interdependent with the maternal system (Mesiano, 2014); the precursor for placental steroid hormones is cholesterol extracted from both maternal and fetal circulation (Tuckey, 2005). Rodent models demonstrate the important role of sex steroid hormones in the onset and maintenance of maternal behaviors (Brunton & Russell, 2008; Numan & Insel, 2003; Saltzman & Maestripieri, 2011). These connections between sex steroid exposures and quality of maternal behaviors also have been repeatedly observed among nonhuman primates, including tamarins, marmosets, titi monkeys, macaques, and baboons, as well as humans (Bardi, Shimizu, Barrett, Borgognini-Tarli, & Huffman, 2003; Fleming, Ruble, et al., 1997; Glynn et al., 2016; Jarcho, Mendoza, & Bales, 2012; Pryce, Abbott, Hodges, & Martin, 1988).

Oxytocin (OT) is produced in the fetal compartment and secreted into the maternal bloodstream (Dawood, Wang, Gupta, & Fuchs, 1978; Malek, Blann, & Mattison, 1996), where it represents approximately 83% of circulating OT (Liu, 2013; Nakazawa, Makino, Iizuka, Kohsaka, & Tsukada, 1984). Few longitudinal studies have assessed OT levels during pregnancy, but most of those have documented some evidence of increases across gestation (Dawood, Ylikorkala, Trivedi, & Fuchs, 1979; De Geest, Thiery, Piron-Possyot, & Vanden Driessche, 1985; Levine, Zagoory-Sharon, Feldman, & Weller, 2007; MacKinnon et al., 2014; Silber, Larsson, & Uvnas-Moberg, 1991). OT is crucially involved in gestational and parturitional processes, but also implicated in a wide range of social and attachment behaviors (Bartz, Zaki, Bolger, & Ochsner, 2011; Campbell, 2010; Heinrichs, von Dawans, & Domes, 2009; Insel, 2010; Kirsch et al., 2005). The role of OT in controlling maternal behavior across species is experimentally evinced by studies in which intraventricular treatment with OT induces maternal behavior in virgin rats (Pedersen & Prange, 1979), intracerebral administration of OT increases approach and touching of infants in macaques (Holman & Goy, 1995), and peripheral administration of an OT antagonist reduces maternal interest in marmosets (Seltzer & Ziegler, 2007). Similarly in humans, women who exhibit higher levels of OT during gestation and the early postpartum period report feeling closer and more attached to their fetuses, display enhanced theory of mind, and exhibit more infant directed gaze, affectionate touch, positive affect, and “motherese” vocalizations (Feldman et al., 2007; MacKinnon et al., 2014).

Corticotropin-releasing hormone (CRH) is a peptide hormone that is synthesized primarily in the paraventricular nucleus of the hypothalamus and plays a central role in regulating pituitary–adrenal function and the physiological response to stress (Vale, Spiess, Rivier, & Rivier, 1981). The placenta, fetal membranes, and decidua all synthesize CRH (McLean & Smith, 1999), but it is placental CRH that is released into maternal circulation, resulting in maternal plasma CRH levels that are 1000 times higher during pregnancy than in the non-pregnant state (Mesiano, 2014). In contrast to the inhibitory influence of maternal stress signals (e.g., cortisol) on expression of the CRH gene in the hypothalamus, maternal cortisol activates the promoter region of the gene in the placenta and stimulates CRH synthesis (King, Smith, & Nicholson, 2001; Scatena & Adler, 1998). Further, placental CRH

stimulates the release of maternal ACTH, which is manufactured from its precursor proopiomelanocortin (POMC) that is partially derived from the placenta (Chen, Chang, Krieger, & Bardin, 1986). Maternal ACTH stimulates cortisol production, which then stimulates placental CRH (pCRH) synthesis and secretion (Mastorakos & Ilias, 2003), resulting in a progressive increase in all three hormones across pregnancy. Although influences of pCRH on the regulation of maternal behaviors have yet to be documented, it is worth noting that through this placental signal the fetus could, hypothetically, manipulate maternal phenotypes indirectly through altering cortisol levels that have been implicated in maternal behavior and attachment in humans and other species (Bardi et al., 2004; Fleming, Steiner, & Corter, 1997; Hennessy, Harney, Smotherman, Coyle, & Levine, 1977; Rees, Panesar, Steinger, & Fleming, 2004).

In addition to the roles of fetoplacental hormones in gestational physiology and onset of maternal psychology and behavior, variations in these hormone levels and trajectories have been implicated in risk for psychopathology. For example, it has been hypothesized that the rapidly declining levels of gonadal steroids may be implicated in postpartum depression (PPD) and postpartum psychosis among vulnerable women (Ahokas, Aito, & Rimon, 2000; Bloch, Daly, & Rubinow, 2003; Bloch et al., 2000; Wieck et al., 1991), midgestation maternal plasma OT concentrations have been associated with PPD (Skrundz, Bolten, Nast, Hellhammer, & Meinschmidt, 2011), and midgestation pCRH has been associated with depressive symptoms both during pregnancy and the postpartum period (Glynn & Sandman, 2014; Rich-Edwards et al., 2008; Yim et al., 2009). These studies exemplify the possibility for fetal tissue-derived signals to be directly implicated in maternal psychopathology.

Fetal behavior

Although rarely considered, one additional pathway of potential fetal signaling is through fetal motor activity. Spontaneous fetal movement transiently stimulates maternal sympathetic arousal (DiPietro et al., 2006; DiPietro, Irizarry, Costigan, & Gurewitsch, 2004). Further, experimentally evoking mild startle response in the fetus, which is characterized by increases motor activity, generates a transient maternal heart rate suppression and an increase in sympathetic activation (DiPietro et al., 2013). The mother does not appear to habituate to the fetal movement stimulus, which continues to invoke a maternal physiological response for the duration of gestation (DiPietro, Costigan, & Voegtline, 2015). The precise biological pathway through which fetal movements might affect maternal arousal is currently unknown. However, it is unlikely that this occurs through conscious perception of these movements. At term, women detect as few as 16% of fetal movements (Johnson, Jordan, & Paine, 1990), which is consistent with the fact that although they are relatively skilled at detecting large or prolonged fetal movements, pregnant women are limited in their ability to detect smaller spontaneous or evoked fetal movements (Kisilevsky, Killen, Muir, & Low, 1991). Given that the pathway likely does not operate through conscious channels, DiPietro et al. (2004) propose that the most plausible mechanism is through perturbations of the uterine wall. They further suggest that these interactions may have broader implications for the role of the fetus in shaping maternal behavior, specifically suggesting that the sympathetic activation in response to the fetal movement signal may begin to prepare the female for the impending demands of

motherhood by redirecting maternal resources away from competing but less relevant environmental demands. These findings and assertions raise the provocative question of whether the degree of prenatal synchrony between mother and fetus might set the stage for postnatal mother–infant interaction.

Fetal microchimerism

In 1979, Herzenberg, Bianchi, Schroder, Cann, and Iverson demonstrated the presence of cells containing a Y chromosome in the plasma of women who were pregnant with male fetuses. Subsequently, cells containing male DNA were demonstrated in the plasma of healthy women decades after giving birth to a son (Bianchi, Zickwolf, Weil, Sylvester, & DeMaria, 1996), a phenomenon described as fetal microchimerism. During pregnancy, there is an asymmetric bidirectional exchange of maternal and fetal cells across the placental barrier (more fetal cells transferred to mother than vice versa; Lo, Lau, Chan, Leung, & Chang, 2000). Fetal cells have been detected in a range of human maternal tissues, including thyroid, heart, liver, lungs, adrenals, kidneys, and bone marrow (Johnson et al., 2001; Khosrotehrani, Johnson, Cha, Salomon, & Bianchi, 2004). A debate exists regarding the purpose of these fetal cells, and whether they exert salutary or detrimental influences on the mother's health and development (Boddy, Forunato, Sayres, & Aktipis, 2015). For example, there is accumulating evidence that they may play a role in maternal wound healing (Mahmood & O'Donoghue, 2014; Nassar et al., 2012), but these cells also have been identified at tumor sites (Kallenbach, Johnson, & Bianchi, 2011) and have been associated with pregnancy complications (Gammill, Aydelotte, Guthrie, Nkwopara, & Nelson, 2013; Gammill, Stephenson, Aydelotte, & Nelson, 2014). Potentially relevant to the issue of maternal programming is the finding that, although the blood–brain barrier usually prevents the passage of cells, during pregnancy it appears that fetal cells may migrate to the maternal brain (Chan et al., 2012). These fetal cells are capable of taking on a range of attributes including neuron-, astrocyte- and oligodendrocyte-like cell types, conceivably allowing participation in neural circuitry and molecular communication (Tan et al., 2005; Zeng et al., 2010). Whether fetal cells in the maternal brain have any functional significance has yet to be demonstrated. Nonetheless, fetal cells are preferentially found in brain regions known to subserve maternal behavior, such as the olfactory bulb (Tan et al., 2005), raising the possibility that the attraction of fetal cells to specific brain areas could represent a pathway through which the fetus affects development and onset of maternal behavior and may have implications for risk for maternal psychopathology (Boddy et al., 2015; Glynn, 2010b).

Evolutionary Perspectives on Maternal Programming

A hallmark of the developmental psychopathology perspective is the emphasis on an interdisciplinary approach. Viewing the maternal–fetal relationship in an evolutionary context can help explain why (and with what ultimate consequence) maternal biology, psychology, and behavior are responsive to fetal signals. Until the 1970s, maternal–fetal biology was generally presumed to reflect a synergistic, cooperative relationship, but Trivers (1974) reframed the parent–child relationship in terms of an evolutionary system of “parent–offspring conflict,” which overturned the peaceful paradigm of maternal–fetal harmony.

Natural selection has shaped the process by which the fetus extracts resources from the mother, and how the mother provides for the fetus, but the evolutionary incentives of a mother and her fetus are slightly misaligned (Parker, Royle, & Hartley, 2002; Schrader & Travis, 2009). This evolutionary tug-of-war is played out, at least mostly, unconsciously, so when we describe mother and offspring “interests” or assign agency to their “strategies,” this is biological shorthand for describing the machinations of natural selection that favor perpetuation of certain allelic variants over others. From the mother’s perspective, the adaptive value of investment (energy, time, and risk) in her pregnancy must be weighed against the costs (energy, attention, nutrition, and risk) to her existing children plus depletion of resources available for her future pregnancies. Meanwhile, selection on fetal traits prioritizes benefits to the fetus itself over the cost of impairing the mother from providing for its siblings and diminishing the likelihood of future siblings being born (Godfray, 1995). In this way, pregnancy becomes a battleground with mother and fetus selected to prefer different amounts of maternal investment, with selection promoting mutual manipulation.

How do the fetus and mother send, interpret, and respond to communication between each other if those messages have been selected to be, sometimes, misleading, threatening, or coercive (Haig, 1996)? Many hormones produced by the fetoplacental unit flow into maternal circulation. A large body of research in evolutionary ecology (particularly ornithology) has investigated how selection shapes the “honesty” of off-spring-to-mother signals (Godfray, 1995; Kilner & Johnstone, 1997). In competitive circumstances with limited resources, an offspring may compensate for her mother’s and siblings’ lower, compared to her own, optimal investment in her by exaggerating her needs (Royle, Hartley, & Parker, 2002). For example, a nestling bird may beg in a way that indicates its need for food is far greater than it really is (Kilner, 1995; Kilner, Noble, & Davies, 1999), because incomplete indulgence of an exaggerated request will (approximately) result in fulfillment of the needed amount. However, from the mother’s point of view, if the fetus’s honest needs cannot be distinguished from dishonest signals, the mother’s best strategy is to ignore all signals (Haig, 1996). The ensuing selective forces can be framed as a game-theory cascade (Bergstrom, 1995) in which fetus preempts anticipated maternal strategy by mimicking signals the mother uses to communicate between different parts of her body that govern the transfer of resources (Haig, 1993), producing hormones biochemically identical (e.g., CRH) or closely resembling (e.g., hPL/luteinizing hormone) nonplacental hormones, which the mother preempts by downregulating maternal production of those hormones (e.g., LH; progesterone) to isolate fetal signals, although the advantage of gaining conveyance between mother and fetus is balanced against the cost of losing conveyance between parts of the mother’s body (Haig, 1996). Ultimately, the fetus is advantaged because placental hormones can “corrupt a mother’s internal lines of communication” (Haig, 1993). Otherwise inexplicable enormous levels of placental hormones in maternal circulation may be due to this arms-race escalation from selection for fetal exploitation of maternal hormone receptors alongside selection for maternal resistance by downregulating receptor expression (Haig, 1993).

The mother’s evolutionarily optimal investment in the off-spring is, theoretically, calibrated to optimize her number of surviving descendants by balancing investment in her own health versus reproduction, and her current versus future pregnancies. This adaptive appraisal takes

into account the mother's somatic resources (physical health and stored energy), number, ages, and quality (health and competence) of her current offspring, and the mother's own age and likelihood to have future offspring (Daly & Wilson, 1995; Williams, 1966). The appraisal is a constantly updating assessment across a woman's reproductive life phase, as well as across the trajectory of any given reproductive event. For example, low-cost (to the mother in terms of time and energy investment) decision points, such as delaying ovulation, are more sensitive to resource scarcity than high-cost decision points, such as spontaneous abortion, which poses a greater health risk to the mother and wastes the time and energy that was already devoted to that pregnancy. Mechanisms of intrinsic monitoring of somatic condition and age may include signals of cellular senescence such as telomere length and/or p53 and Rb protein levels (Ben-Porath & Weinberg, 2005). Mechanisms of extrinsic monitoring of offspring condition and quantity are more difficult to identify but hypothetically could be linked to maternal hormones involved in gestation, lactation, social bonding, or stress.

The maternal–fetal conflict paradigm has broad implications for mental health during (and after) pregnancy. The endocrinology of pregnancy has been implicated in maternal psychological health (Beddoe, Paul Yang, Kennedy, Weiss, & Lee, 2009; Glynn, Davis, & Sandman, 2013), physical health (Magness, 1998; Schlomer, Del Giudice, & Ellis, 2011), cognitive performance (Glynn, 2010b; Henry & Sherwin, 2012), and behavior (Glynn et al., 2016), so fetal signaling, both honest and manipulative, may influence function in these domains. Moreover, the degree to which a mother values investing in her fetus versus current and future offspring varies between mothers and within a mother across her life span. Maternal–fetal conflict decreases as the mother's age increases, because of the inherently diminishing likelihood of future pregnancies (Trivers, 1974). This may result in more harmonious maternal attitudes and health outcomes for later-age mothers.

A woman's energetic investment or even emotional attachment to her fetus may be adaptively calibrated to respond to fetal “vigor,” signals of survival likelihood such as health, strength, or size. The evolutionary cost of diverting time and resources away from current or future offspring and toward a fetus unlikely to survive may result in selection favoring maternal physiologic divestment strategies such as spontaneous abortion (Forbes, 1997; Nepomnaschy et al., 2006; Williams, 1966), behavioral divestment strategies such as limiting the mother's own food intake to feed her existing children more, or emotional divestment strategies. The area of women's mental health where these ideas have received the most attention is PPD (Thornhill & Furlow, 1998). If the mother receives cues during (after) pregnancy of low fetal (neonatal) vigor, emotional and behavioral divestment from that one offspring may strategically enhance a woman's lifetime fertility (number of offspring) by allowing her to shift allocation of resources to instead invest in current or future off-spring with higher likelihood of survivorship (Hagen, 1999, 2002). In this way, selection may have favored for women to respond to signals of poor fetal growth, for instance, with downregulated emotional bonding or even prenatal depression, in preparation for limiting investment in the fetus once it is born (via neglect, or in extreme cases, infanticide (Hagen, 1999, 2002). While these ideas remain controversial, they highlight how viewing maternal psychobiology within the context of evolutionary theory can be useful. The expectation that it is “natural” for women to bond with their fetus/baby and aberrations

from this phenotype represent pathology that could be treated by tapping into some deep, innate instinct is misguided, and may partly explain the lack of efficacy in clinical strategies for preventing or treating antepartum and PPD (Dennis, 2005).

Intergenerational Transmission of Maternal Phenotypes

Understanding developmental transitions requires a consideration of both proximal and distal influences (Lewis, 1999). While this manuscript mostly describes events that occur during a woman's parturient years of life, it is also important to acknowledge the fetal and early life development of a person who will later become a mother. An important characteristic of prenatal programming is that, while some of its influence is evident from the time of birth, it also exerts influence on neuro-psycho-physiologic systems in ways that manifest much later in life. In particular, prenatal programming influences lifelong *responsivity* to both intrinsic and extrinsic conditions, which may only become relevant once the apposite conditions are encountered. Maternal phenotypes appear to be plastic in a way that is sensitive to conditions the mother encountered during her own prenatal and early life development. In this way, prenatal programming and early life experiences of one generation can influence the prenatal programming of the next generation.

Women vary widely in the attributes that are involved in the psychological, behavioral, and physiological traits, proclivities, and abilities involved in motherhood. For example, women are differentially adept at sensing infant needs, communicating with and comforting their infants (Ainsworth, 1979; Meins, Fernyhough, Fradley, & Tuckey, 2001), have variation in postpartum sleep difficulties (Hunter, Rychnovsky, & Yount, 2009), and are differentially physiologically efficient with milk letdown (Neifert, Seacat, & Jobe, 1985). The variance in human maternal phenotypes can be partially ascribed to intergenerational trends (Belsky, Jaffee, Sligo, Woodward, & Silva, 2005; Chen & Kaplan, 2001; Smith & Farrington, 2004; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003). These maternal characteristics may be perpetuated across generations by a dynamic combination of environmental, educational, cultural, and biological influences. Although there is substantial evidence that these continuities can be attributed in part to learning processes, we emphasize that key aspects of maternal phenotypes are plastic and sensitive to early life programming. These include executive function (Buss, Davis, Hobel, & Sandman, 2011), social competence, and affective profiles (Davis, Glynn, Waffarn, & Sandman, 2011; Howland, Sandman, Glynn, Crippen, & Davis, 2016; Lombardo et al., 2012), as well as the physiological mediators of mothering behavior, including stress endocrinology (Davis et al., 2011; Glover, O'Connor, & O'Donnell, 2010), neural circuitry (Babenko, Kovalchuk, & Metz, 2015; Meaney, Szyf, & Seckl, 2007), and neurotransmitter/receptor sensitivity and expression (Herlenius & Lagercrantz, 2004). Evidence from various mammalian species indicates that maternal behavioral phenotypes are plastic, sensitive to early life programming effects, and can be broadcast across generations without alteration to genetic code (Francis, Diorio, Liu, & Meaney, 1999; Gonzalez, Lovic, Ward, Wainwright, & Fleming, 2001; Maestripieri, Lindell, & Higley, 2007). A developmental psychology approach emphasizes these ontogenic, dynamically interacting aspects of psychological development (i.e., an ecological-transactional model of development; Cicchetti & Toth, 1997). The multilevel, developmental programming influences on the psychological, behavioral, and physiological

traits, proclivities, and abilities involved in motherhood exemplify the importance of acknowledging the dynamic interplay between influences and processes across not only an individual's life span but also across generations to understand the origins of maternal phenotypes.

Maternal Programming: Implications for Psychopathology

As discussed above, there is substantial plasticity in neurobiological systems regulating affective, cognitive and social functioning during the perinatal period (Duan, Cosgrove, & Deligiannidis, 2017; Kim, Strathearn, & Swain, 2016; Lonstein, Maguire, Meinschmidt, & Neumann, 2014; Moses-Kolko, Horner, Phillips, Hipwell, & Swain, 2014; Rutherford, Wallace, Laurent, & Mayes, 2015),¹ resulting in heightened vulnerability for psychopathology. Most attention on risk for psychopathology during the transition to motherhood has been directed toward perinatal depression, which affects up to 20% of women (Gavin et al., 2005; O'Hara & McCabe, 2013). Shortly after delivery, up to 85% of women experience a profile of symptoms known as postpartum blues or baby blues, characterized by dysphoric mood, mood fluctuations, crying, anxiety, insomnia, and irritability (O'Hara & McCabe, 2013; O'Hara, Schlechte, Lewis, & Wright, 1991). These subclinical symptoms usually resolve within 10–12 days, unless they develop into PPD (O'Hara & McCabe, 2013). There is some evidence that the early post-partum period is a time of increased risk for major depressive episodes (although a debate exists regarding this issue; Di Florio et al., 2013; O'Hara, Zekoski, Philipps, & Wright, 1990; Vesga-Lopez et al., 2008) and that PPD may be phenotypically distinct from depression occurring outside of the perinatal period. Specifically, sad mood may be less prominent (Bernstein et al., 2008), and anxiety symptoms appear to be more prominent (Hendrick, Altshuler, Strouse, & Grosser, 2000; Ross, Gilbert Evans, Sellers, & Romach, 2003).

Perinatal anxiety disorders are often overlooked (Farr, Dietz, O'Hara, Burley, & Ko, 2014; Goodman, Watson, & Stubbs, 2016; Ross & McLean, 2006), despite that they are frequently comorbid with perinatal depression and may be more prevalent (Matthey, Barnett, Howie, & Kavanagh, 2003; Reck et al., 2008; Ross et al., 2003; Ross & McLean, 2006; Russell, Fawcett, & Mazmanian, 2013). Anxieties during pregnancy and the postpartum period may be uniquely directed toward the health and safety of the fetus/infant or the maternal role/parenting abilities (Fallon, Halford, Bennett, & Harrold, 2016; Martini et al., 2015; Phillips, Sharpe, Matthey, & Charles, 2009). Pregnancy-specific anxiety is defined as anxiety related to maternal and fetal health, labor and delivery, and parenting and has been identified as a separate clinical phenomenon distinct from measures of general anxiety during pregnancy (Blackmore, Gustafsson, Gilchrist, Wyman, & O'Connor, 2016; Dunkel Schetter & Glynn, 2011; Dunkel Schetter & Tanner, 2012; Kane, Dunkel Schetter, Glynn, Hobel, & Sandman, 2014; Misri, Abizadeh, Sanders, & Swift, 2015). The peripartum period is also characterized by increased risk for onset or exacerbation of obsessive-compulsive disorder (OCD), with 40%–80% of women experiencing symptoms at subclinical levels (Maina, Albert, Bogetto, Vaschetto, & Ravizza, 1999; Miller, Hoxha, Wisner, & Gossett, 2015; Misri et al., 2015; Ross & McLean, 2006; Russell et al., 2013; Zambaldi et al., 2009). The most common of

¹Hereafter we refer to the perinatal period as encompassing all of pregnancy and the first postpartum year.

these symptoms are obsessive concerns related to accidentally or intentionally harming the fetus or infant. These intrusive, violent thoughts are more prevalent in peripartum-onset OCD than in OCD without peripartum onset (Uguz, Akman, Kaya, & Cilli, 2007) and, along with other OCD symptoms, are also more common in perinatal depression as compared to nonperinatal depression (Altemus et al., 2012; Wisner, Peindl, Gigliotti, & Hanusa, 1999).

Of the multiple forms of psychopathology for which the peripartum period confers heightened risk, the relative risk for onset and recurrence of bipolar disorder is most pronounced. The relative risk of first-time hospitalization for bipolar disorder is 23 times higher in the first postpartum month as compared to any other phase of the life span (Munk-Olsen, Laursen, Pedersen, Mors, & Mortensen, 2006), and in women known to have bipolar disorder, rates of recurrence are 50%–70% (Viguera et al., 2000, 2007). In addition, women with first onset of unipolar depression during the postpartum period appear to be at higher risk for subsequent conversion to bipolar disorder as compared to women with non-postpartum onset (Munk-Olsen, Laursen, Meltzer-Brody, Mortensen, & Jones, 2012; Sharma et al., 2014). Bipolar disorder can present as postpartum psychosis (Jones & Craddock, 2001), which is considered a psychiatric emergency and is rare, with an estimated prevalence of 1–2 in 1,000 (Sit, Rothschild, & Wisner, 2006). Symptoms of postpartum psychosis include severe sleep disturbances, rapid fluctuations in mood, mood-incongruent delusions, hallucinations, disorganized behavior, obsessions about the infant, and cognitive symptoms like disorientation and confusion (Bergink, Rasgon, & Wisner, 2016; Jones, Chandra, Dazzan, & Howard, 2014; Sit et al., 2006). Psychotic symptoms can also occur at subclinical levels and are more common in women with postpartum-onset depression as compared to pregnancy-onset depression (Altemus et al., 2012).

Consistent with contemporary symptom-based, dimensional approaches to the study of psychiatric disorders (Cuthbert & Insel, 2013), a developmental psychopathology perspective recognizes that psychopathology occurs on a continuum and that contextual factors are critical in defining boundaries between adaptive and maladaptive development along this continuum. For example, somatic symptoms related to sleep, energy, weight, and appetite that are characteristic of psychopathological conditions in non-perinatal phases can result from normative physiological processes of pregnancy and postpartum (Bernstein et al., 2008; Goodman & Dimidjian, 2012; Howard et al., 2014; Matthey & Ross-Hamid, 2011; Misri et al., 2015; Nysten, Williamson, O'Hara, Watson, & Engeldinger, 2013) and may not necessarily indicate psychopathology. Relatedly, symptoms such as heightened anxiety or preoccupation regarding the infant may be a normative feature of new motherhood and represent a healthy maternal response to the infant initially, promoting vigilance, threat detection, and harm avoidance (Leckman et al., 1999, 2004; Wisner et al., 1999). There is evidence of a normative trajectory of these symptoms, which appear to peak in the immediate postpartum period and then begin to diminish by 3–4 months postpartum (Fairbrother & Woody, 2008; Kim, Mayes, Feldman, Leckman, & Swain, 2013; Leckman et al., 1999). Excessive and prolonged worry, or, on the other end of the continuum, an absence of worry, may indicate psychopathology (Kim et al., 2016; Leckman et al., 2004). Furthermore, the content of these early parental preoccupations may determine their course, whereby positive, idealizing thoughts of the infant may promote positive outcomes (Leckman et al., 2004).

Perinatal psychopathology is complex, multiply determined, and heterogeneous in presentation and course. Multiple developmental pathways leading to various manifestations of perinatal psychopathology have been identified and include factors both specific and nonspecific to the peripartum period. Non-peripartum-specific risk factors include a history of psychopathology, low social support, stressful life events, low socioeconomic status, obstetric complications, and personality factors such as insecure attachment style and low self-esteem (Goodman et al., 2016; Goodman & Tully, 2009; Howard et al., 2014; Martini et al., 2015; Milgrom et al., 2008; O'Hara & McCabe, 2013; O'Hara & Wisner, 2014; Paschetta et al., 2014; Robakis et al., 2016; Tebeka, Strat, & Dubertret, 2016; Vesga-Lopez et al., 2008; Yim, Tanner Stapleton, Guardino, Hahn-Holbrook, & Dunkel Schetter, 2015). The most consistently reported perinatal-specific risk factor is an increased sensitivity to the hormone changes of the peripartum period, with both reproductive and stress hormones implicated. Most of this work has focused on PPD (Bloch et al., 2000, 2003; Brummelte & Galea, 2010; Glynn & Sandman, 2014; Rich-Edwards et al., 2008; Skrundz et al., 2011), but there is also evidence for hormone sensitivity in the etiology of perinatal OCD (Labad et al., 2011; McDougle, Barr, Goodman, & Price, 1999) and psychosis (Ahokas et al., 2000; Bergink et al., 2016; Wieck et al., 1991). Other less-studied but promising biomarkers include genetic and epigenetic (Costas et al., 2010; Guintivano, Arad, Gould, Payne, & Kaminsky, 2014; Jones & Craddock, 2007), inflammatory (Bergink et al., 2013; Kendall-Tackett, 2007; Yim et al., 2015), and circadian factors (Lewis, Foster, & Jones, 2016; Sharma, 2003). Multidisciplinary approaches to understanding perinatal risk are warranted and should examine how biological, psychological, and social factors interact to shape developmental trajectories.

The timing of onset of perinatal psychopathology may also determine symptom profiles and course (Di Florio & Meltzer-Brody, 2015; Fisher et al., 2016; Martini et al., 2015; Postpartum Depression: Action Towards and Treatment Consortium, 2015). Perinatal psychopathology that is a recurrence or exacerbation of pre-peripartum symptoms may be distinguished from psychopathology with onset specifically linked to the perinatal period. For example, women with postpartum psychosis usually have one of two disease courses: postpartum psychosis as an extension of bipolar disorder, or isolated post-partum psychosis, with vulnerability only after childbirth (Bergink et al., 2016; Sit et al., 2006; Yonkers et al., 2004). Two distinct expressions of PPD are similarly indicated (Kettunen, Koistinen, & Hintikka, 2014), with those women exhibiting first-onset PPD at higher risk for subsequent PPD episodes as compared to women whose PPD represents a recurrence of previous non-perinatal depression (Cooper & Murray, 1995).

As a sensitive window of development, the perinatal period may also confer opportunities for resilience to psychopathology. Resilience during this life phase may manifest as an absence of psychopathology despite risk, or as competence in the tasks associated with the transition to motherhood in the face of risk (cf. Masten, 2001). While little research has focused on mechanisms of maternal resilience during the peripartum period (Dunkel Schetter, 2011), several studies have identified social support as protective against perinatal depressive symptoms in high-risk women (deCastro, Hinojosa-Ayala, & Hernandez-Prado, 2011; Howell, Mora, DiBonaventura, & Leventhal, 2009; Ritter, Hobfoll, Lavin, Cameron, & Hulsizer, 2000). Further research is needed to identify additional psychological,

biological, social, and cultural factors that promote well-being in vulnerable women (Dunkel Schetter, 2011). Perinatal-specific mechanisms may be particularly interesting and important to consider. Psychological resilience factors such as self-esteem, mastery, and self-efficacy may be enhanced with opportunities for success in achieving the tasks of parenthood and therefore particularly relevant (Howell et al., 2009). It is also plausible that the physiological changes of pregnancy and the postpartum period protect against certain symptoms or conditions. As discussed above, pregnancy and lactation are characterized by downregulated stress responsivity, which may have direct implications for resilience to psychopathology in women at risk. Consistent with this possibility, one investigation observed markedly reduced symptomology in women with bipolar disorder, type I, during pregnancy as compared to before or after pregnancy (Grof et al., 2000). Furthermore, breastfeeding has been prospectively linked to reduced PPD symptoms (Hahn-Holbrook, Haselton, Dunkel Schetter, & Glynn, 2013).

Collectively, existing studies highlight the importance of considering the unique features of the perinatal period that contribute to increased risk for and resilience to psychopathology and inform symptom trajectories. Furthermore, it is clear that there are multiple potential pathways of vulnerability for or protection from perinatal psychopathology. Attention to these distinctive features and differential pathways can advance our understanding not only of perinatal psycho-pathology but also of trajectories of risk and resilience for psychopathology across the life span. Finally, while most research has focused on the consequences of maternal perinatal psychopathology for the developmental trajectories of the offspring, consequences for the developing mother are important to consider in their own right.

Conclusions

A growing body of literature suggests a remarkable neural plasticity associated with reproductive experience. In 1971, Marian Diamond provided a striking example of such plasticity, by demonstrating that the cortical sizes of pregnant rats housed in impoverished conditions matched those of nonpregnant rats housed in enriched conditions (Diamond, Johnson, & Ingram, 1971). For the first time, Diamond's work demonstrated that pregnancy remodels the architecture of the female brain. As with other sensitive developmental periods in the human life span, the transition to motherhood represents an epoch that is well suited for the application of a developmental psychopathology perspective. This is a period in which the context, including the singular embedded relationship with the fetus and the fact that maternal and fetal programming are proceeding in parallel, coupled with the unique developmental tasks requiring mastery by the new mother, result in a significant transformation that is not yet fully understood nor properly characterized in humans. Furthering our understanding of how typical development during this transition proceeds, along with the ways in which trajectories may deviate from the normative, can aid in the understanding of psychopathology and potential interventions. Similarly, understanding how psychopathological states manifest during pregnancy and the postpartum period can inform our understanding of this most fundamental transformation in the female life span.

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